Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
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L2	2182	cobra	US-PGPUB; USPAT	OR	OFF	2004/11/16 09:15
L3	24	1 same 2	US-PGPUB; USPAT	OR	OFF	2004/11/16 09:15
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L5	145	(protease$1 or metalloproteinase$1 or metalloprotease$1) same 4	US-PGPUB; USPAT	OR	OFF	2004/11/16 09:16
L6	14	1 and 5	US-PGPUB; USPAT	OR	OFF	2004/11/16 09:16
L7	10	mocarhagin	US-PGPUB; USPAT	OR	OFF	2004/11/16 09:16
L8	36	3 or 6 or 7	US-PGPUB; USPAT	OR	OFF	2004/11/16 09:17

PGPUB-DOCUMENT-NUMBER: 20040185036

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20040185036 A1

TITLE: Compositions and methods for prolonging survival of platelets

PUBLICATION-DATE: September 23, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Stossel, Thomas P.	Belmont	MA	US	
Hartwig, John H.	Jamaica Plain	MA	US	
Hoffmeister, Karin M.	Cambridge	MA	US	
Clausen, Henrik	Holte		DK	

APPL-NO: 10/ 704377

DATE FILED: November 7, 2003

RELATED-US-APPL-DATA:

non-provisional-of-provisional 60424807 20021108 US

US-CL-CURRENT: 424/94.61, 514/50

ABSTRACT:

The present invention provides modified platelets having a reduced platelet clearance and methods for reducing platelet clearance. Also provided are compositions for the preservation of platelets. The invention also provides methods for making a pharmaceutical composition containing the modified platelets and for administering the pharmaceutical composition to a mammal to mediate hemostasis.

RELATED APPLICATIONS

[0001] This application claims priority under 35 U.S.C. .sctn. 119(e) to U.S. Provisional Application Serial No. 60/424,807, entitled "Compositions and Methods for Prolonging Survival of Platelets," filed on Nov. 8, 2002, which is herein incorporated by reference in its entirety.

---------- KWIC ---------

Brief Description of Drawings Paragraph - DRTX (9):

[0061] FIG. 5 shows GP1b.alpha.-CR3 interaction mediates phagocytosis of chilled human platelets in vitro. FIGS. 5A and 5B show a representative assay result of THP-1 cells incubated with room temperature (RT) (FIG. 5A) or chilled-rewarmed (Cold) platelets (FIG. 5B). CM-Orange-labeled platelets associated with macrophages shift in orange fluorescence up the y axis. The mean percentage of the CM-Orange positive native macrophages incubated with platelets kept at room temperature was normalized to 1. Chilling of platelets increases this shift from .about.4% to 20%. The platelets are predominantly ingested, because they do not dual label with the FITC-conjugated mAb to CD61.

FIG. 5C Undifferentiated (open bars) THP-1 cells express .about.50% less CR3, and ingest half as many chilled-rewarmed platelets. Differentiation (filled bars) of CR3 expression however, had no significant effect on the uptake of RT platelets. Treatment of human platelets with the snake venom metalloprotease, mocarhagin (Moc), which removes the N-terminus of GP1b.alpha. from the surface of human platelets (inset; control: solid line, mocarhagin treated platelets: shaded area), reduced phagocytosis of chilled platelets by .about.98%. Data shown are means.+-.SD of 5 experiments.

Detail Description Paragraph - DETX (45):

[0115] We obtained fluorescein isothiocyanate (FITC)-conjugated annexin V, phycoerythrin (PE)-conjugated anti-human CD1 1b/Mac-1 monoclonal antibodies (mAb), FITC-conjugated anti-mouse and anti-human IgM mAb, FITC-conjugated anti-mouse and anti-human CD62P-FITC mAb from Pharmingen (San Diego, Calif.); FITC-conjugated rat anti-mouse anti-human IgG mAb from Santa Cruz Biotechnology, Inc. (Santa Cruz, Calif.); FITC-conjugated anti-human CD61 mAbs (clone BL-E6) from Accurate Scientific Corp. (Westbury, N.Y.); FITC-conjugated anti-human GPIb.alpha. mAb (clone SZ2) from Immunotech (Marseille, France); and FITC-conjugated polyclonal rabbit anti-vWf antibody from DAKOCytomation (Glostrup, Denmark). We purchased EGTA-acetoxymethylester (AM), Oregon Green coupled fibrinogen from human plasma, CellTracker.TM. Orange CMTMR; CellTracker Green CMFDA, Nile-red (535/575) coupled and carboxylate-modified 1 .mu.m microspheres/FluoSpheres from Molecular Probes, Inc. (Eugene, Oreg.) and .sup.111Indium from NEN Life Science Products (Boston, Mass.). We purchased Cytochalasin B, dimethyl sulfoxide (DMSO), trisodium isothiocyanate (TRITC), human thrombin, prostaglandin E1 (PGE.sub.1), phorbol ester 12-tetradecanoylphorbol-13 acetate (PMA), A23187 ionophore from Sigma (St. Louis, Mo.); botrocetin from Centerchem Inc. (Norwalk, Conn.); and O-sialoglycoprotein-endopeptidase from Cerladane (Hornby, Canada). HBSS containing Ca.sup.2+ and Mg.sup.2+, pH 6.4; RPMI 1640; 0.05% Trypsin-EDTA (0.53 mM) in HBSS without Ca.sup.2+ and Mg.sup.2+; and other supplements (penicillin, streptomycin and fetal bovine serum) were from GIBCO Invitrogen Corp. (Grand Island, N.Y.). TGF-.beta.1 from Oncogene Research Products (Cambridge, Mass.); 1,25-(OH).sub.2 vitamin D3 from Calbiochem (San Diego, Calif.); and Adenosine-5'-Diphosphate (ADP) were from USB (Cleveland, Ohio). Avertin (2,2,2-tribromoethanol) was purchased from Fluka Chemie (Steinheim, Germany). Collagen related peptide (CRP) was synthesized at the Tufts Core Facility, Physiology Dept. (Boston, Mass.) and cross-linked as previously described (Morton et al., 1995). Mocarhagin, a snake venom metalloprotease, was provided by Dr. M. Berndt, Baker Medical Research Institute, Melbourne Victoria 318 1, Australia. Additional unconjugated anti mouse GPIb.alpha. mAbs and a PE-conjugated anti-mouse GPIb.alpha. mAb pOp4 were provided by Dr. B. Nieswandt (Witten/Herdecke University, Wuppertal, Germany). We obtained THP-1 cells from the American Type Culture Collection (Manassas, Va.).

Detail Description Paragraph - DETX (50):

[0120] The N-terminus of GPIb.alpha. was enzymatically removed from the surface of chilled or room temperature maintained and labeled platelets in buffer B, also containing 1 mM Ca.sup.2+ and 10 .mu.g/ml of the snake venom metalloprotease mocarhagin (Ward et al., 1996). After the enzymatic digestion, the platelets were washed by centrifugation with 5.times. volume of buffer A and routinely checked by microscopy for aggregates. GPIb.alpha.-N-terminus removal was monitored by incubating platelet suspensions with 5 .mu.g/ml of FITC-conjugated anti-human GPIb.alpha. (SZ2) mAb for 10 min at room temperature and followed by immediate flow cytometry analysis on a FACScalibur Flow Cytometer (Becton Dickinson Biosciences, San Jose, Calif.). Platelets were gated by forward/side scatter characteristics and 50,000 events acquired.

Detail Description Paragraph - DETX (94):

[0164] Differentiation of human monocytoid THP-1 cells using TGF-.beta.1 and 1,25-(OH).sub.2 Vitamin D3 increases expression of CR3 by .about.2-fold (Simon et al., 1996). Chilling resulted in 3-fold increase of platelet phagocytosis by undifferentiated THP-1 cells and a .about.5-fold increase by differentiated THP-1 cells (FIGS. 5B and 5c), consistent with mediation of platelet uptake by CR3. In contrast, the differentiation of THP-1 cells had no significant effect on the uptake of room temperature stored platelets (FIGS. 5A and 5c). To determine if GPIb.alpha. is the counter receptor for CR3-mediated phagocytosis on chilled human platelets, we used the snake venom metalloprotease mocarhagin, to remove the extracellular domain of GPIb.alpha. (Ward et al., 1996). Removal of human GPIb.alpha. from the surface of human platelets with mocarhagin reduced their phagocytosis after chilling by .about.98% (FIG. 5C).

Detail Description Paragraph - DETX (152):
[0221] Ward, C., Andrews, R., Smith, A. and Berndt, M. (1996). Mocarhagin, a novel cobra venom metalloproteinase, cleaves the platelet von Willebrandt factor receptor glycoprotein Ib.alpha..

Detail Description Paragraph - DETX (176):
[0244] We localized the exposed .beta.-GlcNAc residues mediating .alpha..sub.M.beta..sub.2 lectin domain recognition of GPIb.alpha. N-glycans. The extracellular domain of GPIb.alpha. contains 60% of total platelet carbohydrate content in the form of N- and O-glycosidically linked carbohydrate chain. Accordingly, binding of peroxidase-labeled WGA to GPIb.alpha. is easily detectable in displays of total platelet proteins resolved by SDS-PAGE, demonstrating that GPIb.alpha. contains the bulk of the .beta.-GlcNAc-residues on platelets, and binding of WGA to GPIb.alpha. is observable in GPIb.alpha. immunoprecipitates. UDP-Gal with or without added galactosyltransferase diminishes S-WGA binding to GPIb.alpha., whereas RCA I binding to GPIb.alpha. increases. These findings indicate that galactosylation specifically covers exposed .beta.-GlcNAc residues on GPIb.alpha.. Removal of the N-terminal 282 residues of GPIb.alpha. from human platelet surfaces using the snake venom protease mocarhagin, which inhibited phagocytosis of human platelets by THP-1 cells in vitro, reduces S-WGA binding to chilled platelets nearly equivalent to S-WGA room temperature binding levels. WGA binds predominantly to the N-terminus of GPIb.alpha. released by mocarhagin into platelet supernatant fluids as a polypeptide band of 45 kDa recognizable by the monoclonal antibody SZ2 specific for that domain. The glycans of this domain are N-linked. A small portion of GPIb.alpha. remains intact after mocarhagin treatment, possibly because the open canalicular system of the platelet sequesters it. Peroxidase-conjugated WGA weakly recognizes the residual platelet associated GPIb.alpha. C-terminus after mocarhagin cleavage, identifiable with monoclonal antibody WM23.

Detail Description Paragraph - DETX (178):
[0246] Effects of .beta.-Hexosaminidase (.beta.-Hex) and Mocarhagin (MOC) on FITC-WGA Lectin Binding to Chilled Versus Room Temperature Stored Platelets.

Detail Description Paragraph - DETX (179):
[0247] The enzyme .beta.-hexosaminidase catalyzes the hydrolysis of terminal .beta.-D-N-acetylglucosamine (GlcNAc) and galactosamine (GalNAc) residues from oligosaccharides. To analyze whether removal of GlcNAc residues reduces the binding of WGA to the platelet surface, chilled and room temperature washed human platelets were treated with 100 U/ml .beta.-Hex for 30 min at 37.degree. C. FIG. 11A shows the summary of FITC-WGA binding to the surface of room temperature or chilled platelets obtained by flow cytometry before and after .beta.-hexosaminidase treatment. FITC-WGA binding to chilled platelets was reduced by 85% after removal of GlcNac (n=3). We also checked whether, as exspected, removal of GPIb.alpha. from the platelet surface leads to reduced

WGA-binding after platelet chilling. GPIb.alpha. was removed from the platelet surface using the snake venom mocarhagin (MOC), as described previously (Ward et al, Biochemistry 28, 8326-8336,1996). FIG. 11B shows that GPIb.alpha. removal from the platelet surface reduced FITC-WGA binding to chilled platelets by 75% and had little influence on WGA-binding to GPIb.alpha.-depleted room temperature platelets (n=3). These results indicate that WGA binds mostly to oligosaccharides on GPIb.alpha. after chilling of human platelets, and it is very tempting to speculate that the Mac-1 lectin site also recognizes these exposed sugars on GPIb.alpha. leading to phagocytosis.

US-PAT-NO: 6806360

DOCUMENT-IDENTIFIER: US 6806360 B2

TITLE: Nucleic acids encoding human tissue factor inhibitor

DATE-ISSUED: October 19, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Wun; Tze Chein	Ballwin	MO	N/A	N/A
Kretzmer; Kuniko K.	Wildwood	MO	N/A	N/A
Broze, Jr.; George J.	St. Louis	MO	N/A	N/A

APPL-NO: 10/ 377817

DATE FILED: March 4, 2003

PARENT-CASE:

CROSS-REFERENCE TO RELATED APPLICATION

This application is a continuation of U.S. Ser. No. 09/627,676 filed Jul. 28, 2000, now U.S. Pat. No. 6,534,276, which is a continuation of Ser. No. 09/054,782 filed Apr. 3, 1998, now issued as U.S. Pat. No. 6,171,587, which is a continuation of Ser. No. 08/463,323 filed Jun. 5, 1995, now issued as U.S. Pat. No. 5,849,875, which is a continuation of Ser. No. 08/355,351 filed Dec. 13, 1994, now abandoned, which is a continuation of Ser. No. 08/093,285 filed Jul. 15, 1993, now issued as U.S. Pat. No. 5,466,783, which is a continuation of Ser. No. 07/566,280 filed Aug. 13, 1990, now abandoned, which is a division of Ser. No. 07/123,753, filed Nov. 23, 1987, now issued as U.S. Pat. No. 4,966,852, which is a continuation-in-part of application Ser. No. 07/077,366, filed Jul. 23, 1987, now abandoned.

US-CL-CURRENT: 536/23.5, 435/69.1 , 530/350

ABSTRACT:

A cDNA clone having a base sequence for human tissue factor inhibitor (TFI) has been developed and characterized and the amino acid sequence of the TFI has been determined.

2 Claims, 8 Drawing figures

Exemplary Claim Number: 1

Number of Drawing Sheets: 7

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Detailed Description Text - DETX (8):
FIG. 6 shows an alignment of the basic protease inhibitor domains of TFI with other basic protease inhibitors. All the sequences except TFI were obtained from the National Biomedical Research Foundation Protein Sequence Database (Georgetown University, Washington, D.C., release 13, June 1987). 1. Bovine basic protease inhibitor precursor; 2. Bovine colostrum trypsin

inhibitor; 3. Bovine serum basic protease inhibitor; 4. Edible snail isoinhibitor K; 5. Red sea turtle basic protease inhibitor (only amino acids 1-79 presented); 6. Western sand viper venom basic protease inhibitor I; 7. Ringhals venom basic protease inhibitor II; B. Cape cobra venom basic protease inhibitor II; 9. Russell's viper venom basic protease inhibitor II; 10. Sand viper venom basic protease inhibitor III; 11. Eastern green mamba venom basic protease inhibitor I homolog; 12. Black mamba venom basic protease inhibitor B; 13. Black mamba venom basic protease inhibitor E; 14. Black mamba venom basic protease inhibitor I; 15. Black mamba venom basic protease inhibitor K; 16. .beta.-1-Bungarotoxin B chain (minor); 17. .beta.-1-Bungarotoxin B chain (major); 18. .beta.-2-Bungarotoxin B chain; 19. Horse inter-.alpha.-trypsin inhibitor [amino acids 1-57(1); 58-123 (2)]; 20. Pig inter-.alpha.-trypsin inhibitor [amino acids 1-57(1); 58-123(2)]21. Bovine inter-.alpha.-trypsin inhibitor [amino acids 1-57(1); 58-123(2)]; 22. Human .alpha.-1-microglobulin/inter-.alpha.-trypsin inhibitor precursor [amino acids 227-283(1); 284-352(2)]; 23. TFI amino acids 47-117(1); 118-188(2); 210-280(3)]. Gaps were included in 16, 17, 18 to achieve best alignment. Standard one letter codes for amino acids are used.

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   * * * * * * * * * * * * * STN  Columbus  * * * * * * * * * * * * * *

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ALL COPYRIGHTS AND RESTRICTIONS APPLY.  SEE HELP USAGETERMS FOR DETAILS.

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